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Exhibit 21 A

PRESENTATION
to
THE ROYAL COMMISSION
on
THE WORKMEN'S COMPENSATION ACT

18 October 1966.

Mr. Commissioner:

I have been requested by Counsel for this Commission to attend to-day's hearing, to give evidence concerning silicosis and other chronic disabling lung conditions which may affect men employed in dusty trades, particularly those which entail exposure to silica (i.e., silicon dioxide) dust.

Qualifications

I am a duly qualified physician, on the register of the College of Physicians and Surgeons of Ontario. I hold the Diplomas in Public Health and in Industrial Hygiene granted by the University of Toronto. I am certified as a specialist in Occupational Medicine by the American Board of Preventive Medicine, and am a Fellow of the American College of Preventive Medicine.

For almost twenty years I have been employed in the Industrial Hygiene Branch of the Ontario Department of Health, and for the past two years have been Director of that Branch (Now called the Environmental Health Branch). I am an Associate Professor on the staff of the Department of Physiological Hygiene, School of Hygiene, University of Toronto. Since 1961 I have served as Chairman of the Silicosis Referee Board of the Ontario Workmen's Compensation Board.

I have read the submissions of The Ontario Municipal Association; The Ontario Federation of Labour, The International Union of Mine, Mill & Smelter Workers, and The United Steelworkers of America which raise questions concerning silicosis and the more general problem of disability from chronic respiratory

diseases, particularly emphysema and bronchitis, affecting men exposed to silica dust. The relationship of silicosis to lung cancer has also arisen in the consideration of claims by the Workmen's Compensation Board, and I would ask your Honour's indulgence to speak to this subject as well.

It is necessary, first of all, to define and clarify certain terms. The word "pneumoconiosis" is usually defined as a condition or disease of the lungs caused by the inhalation of mineral or metallic dusts. In general, to reach the lung tissue proper, such dust particles must be less than 5 microns in diameter. (A micron is 1/1,000 of a millimeter, or 1/25,000 of an inch). Larger particles rarely reach the lungs, but are trapped in the nose or mouth, or in the trachea or bronchi, and are later expelled. The reaction of the lung to particles reaching it varies, depending on the chemical or physical properties of the particles, their concentration in the inspired air, etc. Not usually included under the term pneumoconiosis is the acute inflammatory reaction produced by certain metallic dusts such as cadmium oxide or the generalized systemic poisoning which may result from the inhalation of soluble toxic dusts such as lead oxide.

The majority of dusts are inert, in that they produce no acute inflammatory response or chronic fibrosis of the lungs. They may, because of their opacity to x-rays, give rise to shadowing in the chest film. Thus iron oxide deposited in the lung tissue produces a form of pneumoconiosis known as siderosis, characterized by a fine, stippled shadowing throughout both lungs in the chest roentgenograph. This condition is not disabling, and the staining of the lungs by the iron oxide might be compared with the pigmentation of the skin produced by tattooing.

On occasion, respiratory disability may be produced by inert dusts, through a simple over-loading and plugging up of the air sacs with little or no accompanying fibrosis. Cases of such pneumoconiosis have resulted from the

inhakation of talc and nepheline syenite. Both of these minerals are silicates, in which the silicon is combined with one or more other elements such as calcium, magnesium or aluminium. Such cases of pneumoconiosis have occurred rarely, and only under extremely dusty working conditions.

The more disabling forms of pneumoconiosis are caused by the inhalation of dusts which provoke a fibrotic response in the lungs, such as silica or asbestos. (Silica, or silicon dioxide (frequently referred to as "free silica" to distinguish it from the silicates) occurs in nature in the pure form most commonly as quartz. It is the main constituent of sand, and is present in significant amounts in the native rock in the gold and uranium mining areas in this province.

When inhaled over a sufficient period of time, free silica may produce the disease known as silicosis, a condition characterized by multiple small fibrotic nodules scattered throughout both lungs. The nodules may be very small, less than 1 millimetre in diameter, or moderately large, up to 5 or 6 millimetres. Under the microscope they display a characteristic pattern or morphology. The degree of nodulation may vary from sparse to profuse. In his report of 1950, the Honourable Mr. Justice Roach summarized evidence presented by Dr. A. R. Riddell on the subject of silicosis. I would like to read into the record those paragraphs which in my opinion still apply to-day, and to comment on certain statements which for one reason or another should be modified or changed in the light of more recent knowledge.

"There is marked variation between individuals in their susceptibility to the disease. Most people, even in quite marked exposure, are singularly resistant to it. On the other hand there are some persons who seem to be peculiarly susceptible to the disease.

"The industries in which the workers are subject to the disease are those in which the workers are likely to inhale excessive quantities of silica dust. They include mining, sand-blasting, porcelain making, rock grinding, stone



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cutting, moulding and the grinding of materials which are grimy with sand." By way of comment, to these industries might be added the manufacture and use of silica brick, as in the steel-making industry, the spraying of vitreous enamels, and the making of certain abrasive cleansers. ///

To quote again: "The length of exposure sufficient to cause the disease in its compensable form varies in different industries and varies also in the individuals. Taking the average worker in the various industries it would appear that miners and quarry workers may develop it in 13 to 20 years; porcelain workers in certain occupations in 14 to 20 years; granite cutters in 27 to 29 years; moulders in 28 to 30 years"

"A worker may develop a certain degree of silicosis, that is a certain amount of fibrous tissue in the lungs, and, notwithstanding continued exposure, the condition may remain stationary". To this might be added the converse statement that progression of the condition may continue, even though the worker has been removed from exposure. Similarly, silicosis may first become apparent in the chest x-ray several years or more after the worker has left exposure.

Again I quote: "The disease occurs in two forms. One is termed simple silicosis, that is the physical state which is marked only by a fibrotic condition of the lung tissue. The other is termed complicated silicosis, that is the physical state in which the fibrotic condition is complicated with infection, mainly tuberculosis of the lung.

"Simple silicosis may vary in extent from the stage where the number of nodules are few and small to the stage where large areas of the lungs are involved". As the condition progresses, nodules which lie close together tend to fuse or coalesce, and thus larger areas of the lung tissue become fibrosed.

"Simple silicosis renders the lung less resistant to tuberculosis. Since tuberculosis is caused by infection it is imperative that a workman suffering from simple silicosis should not be subjected to the danger of such infection. Not only is the silicotic susceptible to tuberculosis but the tuberculant is susceptible to silicosis. Therefore a person who has tuberculosis or has had it but been cured should never be exposed to the inhalation of silica dust".

In my opinion, the last statement should be clarified. It is correct if the reference to tuberculosis is interpreted as meaning active tuberculous disease or arrested pulmonary tuberculosis which has left evidence of scarring of the lungs. There is ample evidence, both experimental and human, to indicate that such persons are poor risks for work in silica exposure. On the other hand, I know of no valid evidence to indicate that a persons is a poor risk for silica exposure who has a clear chest film and has only a positive tuberculin test to show that he has had a previous infection with the tubercle bacillus.

During the years prior to 1950, tuberculosis, as stated by Dr. Riddell in the sentence which followed the above quotation, accounted for 60 to 70 percent of the deaths among silicotics. At that time pneumonia and right heart failure, both of which might be considered complications of silicosis, were responsible for a further 10 to 15 percent of deaths in silicotics. With the advent of chemotherapy for tuberculosis, this picture has changed radically. During the period 1960 to 1964, tuberculosis was responsible for only about 8 percent of the deaths in silicotics, with pneumonia accounting for approximately 20 percent and right heart failure for about 24 percent. Nearly 48 percent of silicotics now die of conditions unrelated to their silicosis, such as coronary artery disease, cancer, cerebral haemorrhage, cancer, accidents, etc. This changing mortality picture has undoubtedly resulted in the rejection of a relatively higher proportion of claims for death benefits and pension benefits to widows and dependents, and has almost certainly generated feelings of dissatisfaction over the settlement of specific claims. Such feelings are understandable, and

In older age group
more
have
post-tuberculous

Right side primary
Back up primary

I have every sympathy for the widow so deprived of a pension. However, such settlements must be based on whether there is medical evidence or expert opinion relating the cause of death to the occupational disease from which the worker suffered. If there is none, then the allowance of benefits, in my opinion, would constitute a misuse of compensation funds for purely social or welfare purposes.

It is perhaps appropriate here to consider the question of lung cancer occurring in a silicotic. In the majority of such cases, the lung cancer will probably be the eventual primary cause of death. The basis question is, "Did the silicosis cause the lung cancer to develop?".

Lung cancer presently accounts for some 6 percent of deaths in males over the age of 60 years in Ontario, regardless of what occupations they have worked in all of their lives. Since 1961 there have been 355 deaths from all causes among silicotics in Ontario, of which 21 were due to lung cancer. Dr. A. H. Sellers, former Director of the Medical Statistics Branch, Ontario Department of Health, has computed the number of lung cancers which would normally have been expected among these 355 silicotics if they had sustained the same proportion of lung cancer deaths in each age group as occurred in Ontario males. The number expected was estimated at 16, compared with the 21 cases which occurred. While this represents an increase of some 30 percent over the number normally expected, the increase is not statistically significant. To express it crudely, there was about a fifty-fifty chance, when 16 cases were normally expected, of having as many as 21 cases occur.

An increase of 30 percent over the normal lung cancer expectancy in this group of men may be compared with the findings in other studies of lung cancer in relation to occupation. In five such studies which I have either carried out or assisted in, it was found that the risk of lung cancer was roughly 5 to 7 times normal in certain specific occupations. Increases of this magnitude would occur by chance less than 1 in 100 or 1 in 1,000 times. As a result of

these investigations it was considered that the increased risk of lung cancer was related to occupational exposure, and in the three studies done in Ontario lung cancer was accepted as a compensable condition when occurring in the specified occupations and action was taken to eliminate the hazard at the work-places.

Increases of the same order of magnitude have been reported for lung cancer in at least two industrial studies performed in the United States, two in Great Britain, two in Europe, and one in Japan. In none of the studies mentioned was exposure to silica considered to be related to the increase. In several of the studies where mining was involved, radio-active gasses and dusts were also present in the atmosphere in high concentrations, and the increase in lung cancer was considered to be associated with this exposure.

In South Africa, where there has been longer experience with silicosis than we have had in Ontario, no increase in the incidence of lung cancer has been found, over what might normally have been expected.

From the experimental side, it might be noted that no cases of lung cancer have been reported in the thousands of animals which have been used over the past forty or fifty years in studies of silicosis.

It is therefore my considered opinion that there is no evidence to indicate that silicosis or exposure to silica dust results in an increased risk of developing lung cancer. This should not be taken to imply that the subject is closed. The experience of our miners, and of our silicotics in particular, is under continuing review.

The foregoing conclusion concerning the relationship between silicosis and lung cancer is in contrast to the known experience in cases of asbestos fibrosis of the lungs. Various studies have shown that lung cancer has been responsible for from 15 to 23 percent of all deaths occurring in men with asbestosis. Investigations are under way to determine whether the increased

risk of lung cancer is associated with all kinds of asbestos, or restricted to fibres of a certain type. Lung cancer has been accepted as a compensable condition in the few cases of asbestosis in Ontario in which it has developed.

Chronic bronchitis is a clinical disorder characterized by excessive mucus secretion in the bronchial tree, accompanied by chronic or recurrent cough. The condition is most prevalent among males over 35 years of age, and occurs predominantly, but not exclusively, in cigarette smokers. There are no characteristic abnormalities seen in the chest x-ray film. Any alterations of respiratory function consist primarily of ^{increased} ~~increased~~ resistance to air flow.

1 Emphysema is an anatomic alteration of the lung characterized by an abnormal enlargement of the air spaces distal to the terminal bronchioles and accompanied by destructive changes in the alveolar walls. In lay terms, there is over-distension and destruction of the walls of the small air sacs at the end of the bronchial tree. The tissue changes may vary in their distribution and extent. Thus the emphysema may be paracicatricial, that is, an over-distension of the air spaces and destruction of the alveolar wall in those areas of lung adjacent to fibrotic lesions of the lung.

2 In lobular emphysema, the over-distension and destruction of the walls of the air sacs occurs in anatomic segments of the lung known as secondary lobules. This form of emphysema is not associated with fibrotic lesions. It may involve one or more lobes of the lungs, or may be generalized, affecting all areas of both lungs. Obstruction to air flow is a common feature of lobular emphysema.

Closely allied to lobular emphysema is the focal emphysema first described by Gough in men with coal-workers' pneumocniosis in Great Britain. In this condition there is dilatation and distension of the air spaces in the lobules, but destruction of the alveolar walls is not a feature nor is obstruction to the respiratory air flow common. According to Gough, focal emphysema character-

istically develops in men exposed to coal dust. It may also develop following inhalation of natural graphite and pure carbon dusts.

3 Another form of emphysema, known as generalized vesicular emphysema, involves the lung as a whole rather than affecting the lobules individually. In this form of emphysema there is relatively uniform enlargement of the air spaces diffusely throughout both lungs, but little destruction of the walls of the air sacs unless the condition is severe

Clinically, some persons with emphysema have no symptoms or abnormal physical signs, and the chest film may be normal even though emphysema is found on pathological examination of the lungs. Where the disease is more marked patients will usually complain of shortness of breath on exertion, and when severe, air hunger may occur even at rest. The diagnosis is usually made clinically and by pulmonary function studies, the chest x-ray providing confirmatory evidence. Emphysema usually occurs in men over 40 years of age who have a long history of chronic bronchitis and cigarette smoking. In advanced cases, the disease is totally disabling.

In Great Britain, for many decades, chronic bronchitis has been a major cause of incapacity for work, and the disease has been the subject of many studies. In the United States and Canada, mortality data are available only for the years since 1950, approximately. In both countries the crude death rate from emphysema, with or without mention of chronic bronchitis, has risen faster than other causes of death, including lung cancer, during the past fifteen years. More complete studies in the United States have demonstrated that the death rate for emphysema in white males rose more than 6 times from 1949 to 1959. A recent survey by Dr. A. H. Sellers of Ontario mortality during the years 1951 to 1963 has revealed that the greatest increase in emphysema has occurred in men over the age of 55 years. Among men aged 55 to 64 the death rate

tripled; among those aged 65 to 74 the increase was nearly fourfold; and in men aged 75 and over the increase was more than 8 times.

It is evident that with an increasing incidence of emphysema in the older male age groups generally, more and more workers in dusty trades will also develop the condition. The question before us is whether exposure to dust over many years will result in an even greater increase in the disease in these workers.

The cause of emphysema has been the subject of considerable research particularly in the past decade. With reference to the lobular and generalized vesicular forms of the disease, the general concensus of reports to date is that cigarette smoking is a major factor in their development, and that air pollution also plays a part, though a lesser one. Focal emphysema is a recognized result of the inhalation of carbonaceous dusts. Advanced cases of simple silicosis may occur without any evidence of emphysema. However, emphysema can occur around silicotic nodules, though with less frequency than in coal-miners' pneumoconiosis. In cases of silicosis wherein there has been formation of large fibrous masses, localized areas of paracicatricial emphysema frequently develop. In such cases, the emphysema is recognized as an integral part of the silicotic process, and insofar as the emphysema increases the patient's disability it is considered compensable.

There is as yet no valid evidence to show that the common forms of emphysema, the lobular and the generalized vesicular forms, occur with greater than normal frequency in men exposed to silica. This subject also, however, is under continuing study, in this country and in others.

Before closing, I would like to draw to the attention of this Commission the way in which claims for silicosis are processed, since it is quite different from the manner in which claims for other industrial diseases are handled. Provided that the silicosis claimant can show that he has had two years of silica exposure in Ontario, and that he has not subsequently sustained two years or more of silica exposure elsewhere, he will be referred to the Silicosis Referee Board for examination prior to consideration of his claim. The Referee Board is asked to give its opinion as to whether or not silicosis is present and to assess the degree of disability therefrom. In other words, the claimant is examined by impartial referees who have been selected as experts in the field of silicosis, before the initial decision is given on a claim. This is quite the reverse of the procedure followed with other industrial injuries. Before rendering its opinion, the Silicosis Referee Board recognizes that it must have as much information as it is possible to obtain on the case. Not infrequently, in difficult cases the Referee Board will ask for special investigation in hospital or sanatorium before rendering its opinion. The Referee Board also stands ready to reconsider its opinion at any time in the light of new medical information. Once seen by the Referee Board, the individual case is customarily followed, at intervals ranging from 6 months to two or three years, so that further developments, such as increasing disability, can be assessed.

It is therefore respectfully suggested to this Commission that the medical opinions of the Silicosis Referee Board should not be subject to reversal by decisions of the Review Committee, the Appeal Tribunal or the Compensation Board itself, particularly when no new medical evidence is submitted at these hearings. If such evidence is forthcoming, the case should be referred back to the Silicosis Referee Board for reconsideration. It is, of course, quite proper that the appeal framework should review the decisions of the Referee Board to ensure that that body has, in fact, obtained and reviewed all pertinent information

available in a case before it renders its opinion. To ensure both management and labour that fair, impartial and expert consideration was given to each case, thought might be given to having a physician designated by each side attend Referee Board meetings as observers. 24

The basic point is that the diagnosis of an occupational disease such as silicosis, the assessment of physical disability, the relationship of the disease to other conditions which may develop concurrently, are all matters to be decided essentially on medical grounds, rather than legalistically.

